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A Thesis on
Post-Anaesthetic Necrosis

with reference to the
Puerperal State.

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POST-ANAESTHETIC ACETONURIA
with reference to the PUERPERAL STATE.

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The following fourteen cases of Child Labour; in which Chloroform was the anaesthetic used, occurred in general practice.

The investigation of these cases was prompted by the first case of the series, in which certain alarming phenomena suggested the probability of Delayed Chloroform Poisoning as being the cause of the very grave signs and symptoms which presented themselves.

An attempt was made to determine the following points, with reference to Child Labour in which Chloroform Anaesthesia was employed.

(1) Whether Acetonuria was present before, and after the labour.

(2) Whether any pre-existing condition of the Digestive Circulatory, Respiratory or Renal Systems was present which might pre-dispose to Chloroform Poisoning.

(3) Whether any signs of poisoning supervened which might be directly attributable to the Chloroform Anaesthesia.

(4) Whether any early train of symptoms existed which might help to make the diagnosis of Chloroform Poisoning distinct from Sepsis.

(5) Whether any early train of symptoms existed which might indicate Ant-Acid treatment.

THE ACETONE BODIES.

Before entering into the discussion of the cases of Child Labour shewing Post Anaesthetic Acetonuria, the question arises what are the Acetone Bodies, and what is their Clinical significance.

The Acetone bodies are :-

- (1) B-Oxybutyric Acid, which by oxidation becomes
- (2) Diacetic Acid, and this again by loss of $C.O_2$ becomes
- (3) Acetone.

Acetone is stated to be present as the faintest trace in the Urine of healthy persons: but Von Jaksch¹ and V Noorden² hold that the presence of B-Oxybutyric Acid and Diacetic Acid in the Urine is pathological.

Further, the Acetone bodies may be present in the Urine of such conditions as Starvation, Digestive disorders, Diabetes, Fevers; and after the administration of inorganic poisons such as Phosphorus and Phloridzin etc: under the classification of poisons are grouped the so called Post Anaesthetic cases following the administration of Chloroform, Ether, Ethyl Chloride, or Nitrous Oxide.

A distinction has been drawn by Guthrie³ between Acidosis and Fatty Acid Intoxication: the terms have been, and are used to distinguish the condition in which certain organic acids occur in abnormal amount in the blood and urine (Acidosis), from one in which toxic symptoms are added to their presence (Acid Intoxication).

The immediate precursor of Acetone is a Fatty Acid: what then is the source of the fatty acid?

Formerly the origin of the Acetone bodies was considered to be from Carbohydrate.

This view has been discarded.

Their origin has been claimed to be from Proteid: but Langdon Brown⁴ has pointed out that the excretion of acetone is not accompanied by so great an excretion of Sulphur and Nitrogen as would be the case if the acetone bodies were derived from proteid alone.

Geelmuyden⁵ has produced acetonuria in a healthy adult, by means of an exclusive diet of fat: in a few days B-oxybutyric Acid, Diacetic Acid, and Acetone were found in the Urine in large quantities.

The general opinion now held is that the acetone bodies are chiefly derived from fat, though they may be originated from proteid to a limited extent.

THE CLINICAL SIGNIFICANCE OF THE ACETONE BODIES.

The signs and symptoms observed during a fatal case of Delayed Chloroform Poisoning are fairly typical of any other variety of Acid Intoxication.

The clinical picture is somewhat as follows:- 12 hours or so after the anaesthetic, sometimes more sometimes less, the patient vomits repeatedly, the vomit may eventually be haemorrhagic, and jaundice may be present: there may be restlessness and delirium, the smell of acetone may be noted in the breath and the acetone bodies may be present in the urine: this is followed by drowsiness, apathy, and unconsciousness, deepening into coma, which is terminated sooner or later by death.

Many theories have been advanced in order to explain the toxic phenomena of acid intoxication, occurring in post anaesthetic cases, acute yellow atrophy of the liver, and in fact in all cases imputed to intoxication by acetone bodies.

The fatty acids themselves are not the direct cause of the toxaemia, for if fatty acids be given to man or animals they do not even produce acetonuria.

As yet no completely satisfactory explanation is forthcoming.

In all fatal cases of Delayed Chloroform Poisoning the Necropsy shews there to be fatty degeneration of the liver, Kidneys, and other organs to a less extent.

An explanation of this is given by Stiles and MacDonald⁶ and later by Carmichael and Beattie⁷: the former hold that the anaesthetic itself is the primary cause of the fatty degeneration: they have proved experimentally that chloroform causes fatty changes in the liver and kidneys; and further they have shewn Chloroform to be far more detrimental in its toxic action on the tissues than Ether.

Again these two observers have suggested that Idiosyncrasy plays an important part in the re-action of the patient to chloroform, just as some individuals are more susceptible to the action of a certain drug than others are, which last is a well known fact.

Beesly⁸ has apparently proved the following statement in his admirable paper, which strengthens the above views of Stiles and MacDonald, namely:- that there is no relation between Delayed acetone excretion, duration of operation, nor amount of anaesthetic administered.

On the other hand Guthrie holds, that in fatal cases of Delayed Chloroform poisoning, the anaesthetic is merely "the last straw", causing a fatal result by acting on a previously fatty liver.

It is not intended here to enter into a further discussion on the merits of either theory, suffice it to say that the whole question of acid intoxication is as yet undecided. But besides the fatal cases of Delayed Chloroform Poisoning, all degrees of intensity may be met with down to those cases in which the symptoms are so slight that the condition is liable to be overlooked.

Stiles and MacDonald⁶ emphasise the fact that cases shewing symptoms of chloroform poisoning, albeit of slight degree only, have frequently been mis-interpreted the toxic phenomena being attributed to delayed shock or sepsis.

CHLOROFORM ANAESTHESIA WITH REFERENCE TO PARTURITION.

That the parturient woman enjoys an immunity to chloroform is an established fact, which is at present unaccountable.

Blumreich⁹, in 1899, apparently demonstrated a considerable increase in the alkalinity of the blood in pregnancy, both for human beings and also for rabbits. From this it might be argued that the blood of a parturient woman would be more able to neutralise the acid products of metabolism due to chloroform, than the blood of a non-parturient woman. But this

is pure hypothesis, as the reaction of the blood in post anaesthetic cases after operation, and after parturition has yet to be worked out: and also the metabolic changes in the tissues caused by the anaesthetic, and their reference to the presence of the acetone bodies are as yet not determined. Further, haematologists are not yet agreed as to what the reaction of normal blood is, Van Noorden, Friedenthal and others maintaining that it is neutral.

Whatever be the cause of this immunity to Chloroform the fact remains that there has been no death recorded, occurring during the parturition of a healthy woman, which could be directly attributed to the toxic action of the chloroform alone. In fact we are brought up as Medical Practitioners to consider the administration of chloroform to parturient women as being so safe that we consequently never apprehend any untoward result from its reasonable use.

But occasionally a case such as the following is experienced :- The patient exhibits some "malaise" perhaps for the first two days of the puerperium, possibly complaining of headache with or without nausea, and accompanied on rare occasions by vomiting: the labour may have been simple, with no instrumental interference, obstetrical anaesthesia having been

administered, the degree of narcosis being most intense during the passage of the head over the perinaeum.

The symptoms associated with the secretion of milk, or the so called "Milk Fever", are quite distinct from the "malaise" described.

It is a well known fact to many that such a condition is occasionally noted after a labour which has not necessarily been severe, and the explanation usually given is that the "shock of the labour" is responsible for it, or possibly some "mild septic change".

Beesly⁸ has shewn that acetonuria is present in practically all post operative cases after a general anaesthetic, sometimes associated with toxic symptoms of varying degree, sometimes no toxic affects are noted at all. Therefore it is reasonable to infer that acetonuria may be present in the puerperium after a labour in which chloroform was administered, and that the patient may occasionally shew signs of poisoning in varying degree, just as post operative cases do.

If the increased alkalinity of the blood is the key note to a parturient woman's immunity to Chloroform, any morbid condition such as an auto-intoxication e.g. from obstinate and prolonged constipation might tend to diminish that alkalinity; then if the toxæmia should

persist, the patient come to term and be delivered under chloroform anaesthesia, it is reasonable to expect that there might be signs of acid intoxication, that is possibly headache and nausea or vomiting during the first day or so of the puerperium, with concomitant acetonuria.

In all the cases examined by the writer, in which chloroform had been administered during the labour, acetonuria was present.

The first case, which is here given in full, shewed somewhat alarming phenomena, in addition to the presence of Diacetic Acid and Acetone in the Urine.

C A S E I.

Mrs T. Primipara: aet 28.

A small woman about 5ft 1in in height, of a sallow complexion and dark hair.

She gave the impression that her mind had been developed to the detriment of her body".

FAMILY HISTORY. Rheumatism on both sides: the patient said her parents were "highly strung and nervous people".

PREVIOUS HEALTH. As a rule good: occasional attacks of constipation more troublesome during the last two or three years: she sometimes suffers from Dyspepsia. Circulatory and Respiratory Systems - shewed no signs of organic disease as evidenced by examination.

A year before the birth of her child when the Abdomen was examined no area of tenderness could be palpated: the fundus of the stomach extended to just above the umbilicus.

The upper margin of the Liver was at the fifth rib

the lower margin two fingersbreadths below the costal margin in the mammary line.

There was no previous history of any general anaesthetic having been administered.

THE PREGNANCY: was uneventful except for occasional attacks of obstinate constipation especially marked during the last fortnight when it yielded to appropriate treatment.

The Urine shewed on three occasions an average result of:- Specific Gravity 1015: Acid: no Sugar: no Albumin: nor tube Casts.

Five weeks before the onset of labour the liver dulness was apparently unchanged.

THE LABOUR: proved to be protracted and abnormal, owing to the premature rupture of the membranes, and escape of the liquor amnii, during the early part of the first stage (approximately of 30 hours duration). The bladder and rectum were carefully attended to throughout the labour. During the second stage slight chloroform anaesthesia was maintained during the pains. Eventually a living child was delivered by forceps, the perinaeum being torn; the tear did not extend into the rectum. The third stage lasted for an hour and a half. The placenta, which was entirely adherent, had to be removed by the gloved hand under chloroform; a hot douche was then given, and ergotin 1/100th grain injected hypodermically into the buttock.

The condition of the patient proving satisfactory, full anaesthesia was again maintained and the perinaeum repaired. Five and a half ounces of chloroform were used, given on a schimmelbusch mask, which allows of free evaporation. The periods of anaesthesia during the second and third stages and subsequently, gave an approximate total of four hours.

PUERPERIUM.

FIRST DAY. The patient had slept almost continuously from the time when chloroform was withheld: the uterus was well contracted; the pulse was 90 and the temperature 99° F. The urine was drawn off by catheter. It was of a dark amber colour, with a pungent aromatic odour. The patient took nourishment well during the day: she complained of slight headache and drowsiness in the evening. Fifteen ounces of urine were obtained by catheter.

SECOND DAY. After a fair night, she said she "felt well and ready for food". At 9 a.m. the pulse was 100 and the temperature 99° F. There was slight general jaundice, most marked on the malar prominences and the sides of the nose. The skin was dry. The liver dulness was found on percussion to be unchanged.

The uterus was well contracted, the lochial discharge not offensive, and the perineal wound healthy. The lochial discharges and perineal wound progressed to a favourable issue, and no septic changes were noted at any time.

Fourteen ounces of urine were obtained by catheter (after this it was passed naturally): See Urine Chart page .

At 3 p.m. she was restless and drowsy, complaining of hiccough, thirst, frontal headache, and nausea, though there was no vomiting. Pulse varied from 105 to 110, temperature 99.4° F., respirations 30. Calomel (gr. 5) was ordered - every hour one grain.

By 10 p.m. her expression was anxious. She could not sleep: the hiccough was relieved by teaspoonfuls of champagne. She frequently asked for drinks, but only took nourishment after persuasion. Pulse 110-116 Temperature 99.4° F: respirations 34. She passed so restless a night that the nurse tied her thighs together to protect the perineal stitches.

THIRD DAY. At 9 a.m. her pulse was 120 regular, of small volume; temperature 100° F; respirations 32. She complained of drowsiness and of a sweet taste in her mouth. The jaundice was of a deeper colour, especially marked on the face and in the conjunctivae. Her expression was anxious, her pupils dilated; the tongue was furred, its tip being denuded of epithelium; the breath had an ethereal odour, with a smell of apples; no perspiration was noted.

She was encouraged to drink water, milk, and barley-water; elimination by the skin was attempted by blankets and hot bottles. Tincture of Digitalis (minims 5) was ordered every four hours, and Sodium Bicarbonate grains 30 with Aqua Menthae Piperitae to the ounce, every two hours. The champagne was withheld, whisky (half a ounce) and one teaspoonful of Puro in water being substituted every four hours.

At 3 p.m. the bowels acted freely; after enemata of hot water, about ten ounces of Sodium Bicarbonate solution (two ounces to the pint of hot water) were left in the bowel.

At 10 p.m. she was delirious, the acetone smell being strong in the breath. Pulse 120; temperature 100.4° F; respirations 39. Thirty five ounces of urine were collected since the previous midnight; specific gravity, 1028, brown amber colour, markedly acid; no albumin; acetone and diacetic acid present. The deposit shewed uric acid and urates, and some crystals like tyrosin, but too small.

FOURTH DAY. At 9 a.m. she had passed a restless night with marked delirium; vomiting began at 11 p.m. consisting of bile, mucus, and the food taken.

The pupils were widely dilated, the breath charged with acetone; the skin was dry, the jaundice being unchanged. Pulse 137; temperature 100.8° F., respirations 46.

Copious enemata was ordered every six hours, with as much Sodium Bicarbonate solution to be left in the bowel as possible; during this procedure great care was taken of the perineal wound. A mixture containing Sodium Bicarbonate grain 60, Sodium Citrate grains 20 was ordered every two hours.

At first everything was vomited up, but either by repeating the dose or by making the patient drink freely of water, which usually resulted in her vomiting, the soda mixture was retained. In the afternoon the vomiting occurred about once every two hours.

At 6 p.m. she could retain her medicine, and also teaspoonfuls of puro and water.

At 10 p.m. she had vomited once since 6 p.m. She was rational, but had very confused ideas as to what had happened. Calomel (grains 3) was ordered. Pulse 115; temperature 100° F.; respirations 40. At midnight she was perspiring freely, and inclined to sleep. Fifty ounces of urine were collected, specific gravity 1026, acid; acetone present. A few uric acid crystals and abundant urates were deposited.

FIFTH DAY. She was much improved, there had been no vomiting, and she had taken nourishment well during the night.

At 9 a.m. her pulse was 100, of a larger volume, temperature 98.6° F.; the jaundice was not so marked. The pupils were of normal size, and no odour of acetone was noted in the breath.

The Sodium Bicarbonate was given every four hours; the enemata were stopped when the bowels moved; the whisky and digitalis were discontinued.

At night her pulse was 90; temperature normal. She had taken nourishment well during the day and had not

vomited; One hundred ounces of urine were collected of a straw colour, specific gravity 1018, moderately acid, acetone present and a deposit of urates.

SIXTH DAY. Her condition shewed further improvement. The jaundice had faded markedly. The Sodium Bicarbonate was reduced to grains 40 every four hours, without the Sodium Citrate; Eighty ounces of urine were collected, still acid, and containing acetone.

From this she steadily progressed to recovery; the urine was slightly acid and contained acetone till the eighth day when it was alkaline with no acetone. The Sodium Bicarbonate was discontinued on that date.

URINE. CASE I.

For the tests employed see page

The average of three Analyses of the Urine before Labour gave:- Specific Gravity 1015: Acid: No Albumin no Sugar: nor Casts.

CHART OF URINE DURING PUERPERIUM.

"P" = Puerperium: "1", "2", etc, = Days: "S.G." = Specific Gravity: "R" = Reaction: "U" = Urea: "A" = Daily Amount of Urine in ounces: "C" = Catheter: "?" = the quantity was doubtful: "B.C" = Blood Corpuscles in the deposit.

P.	S.G.	R.	Acetone	Sugar	Deposit	U.	A.
1			Not tested.				C 25 ?
2	1029	Acid very.	Not tested	Nil.	Uric Acid. Urates. No Casts.		C 14 ?
3	1028	Acid very.	Present also Diacetic Acid.	Nil.	ditto Tyrosin ? B.C.		30
4	1024	Acid	Present.No Diacetic Acid.	Nil.	ditto No Tyrosin		51
5	1018	Acid	ditto.	Nil.	ditto		100 ?
6	1019	Acid Slight	ditto	Nil.	Slight Urates B.C.		80 ?
7	1016	Acid Slight	ditto.	Nil.	-		-
8	1016	Alkal- ine.	No Acetone	Nil	-		?

There was no Albumin present in the Urine of the second day: which was obtained by Catheter.

Albumin has no "place" on the Charts of Puerperal Urine: it is noted if it was present or not in Catheter Specimens.

The amount of Urea was not estimated.

The regular descent of the Specific Gravity from 1029 -1016 should be noted: likewise the Reaction, which was at first very Acid finally becoming Alkaline.

Diacetic Acid was found on one occasion but it was never demonstrated afterwards.

The reaction for Acetone on the third, fourth, and fifth days was obtained by the Sodium Nitroprussiate test; the colour given was a deep "blackberry" purple, the foam being similarly tinged; on the sixth and seventh day the colour of the reaction had faded most markedly.

In the deposit on the third day, among the "sheaves and needles" of Uric Acid, certain small crystals were observed very similar to Tyrosin but smaller and of a pale yellow colour; no Leucin crystals were noted.

In a reported case of Acute Yellow Atrophy of the Liver by de Havilland Hall¹⁰ similar crystals were found in the Urine; they were said to be identical with Tyrosin but too small in size.

DISCUSSION OF CASE I.

The Clinical signs exhibited in the Puerperium of this case are very similar to those present in the cases of Post Anaesthetic Acetonuria with Delayed Chloroform Poisoning, and Acid-Autointoxication as recorded by Beesly⁸, Brackett Stone and Low¹¹, Hubbard¹², Morse¹³, Stiles and MacDonald⁵, and others.

But before such a diagnosis is arrived at, it is necessary to emphasise, and recapitulate certain points in the history; and also to discuss various conditions which might give rise to similar clinical phenomena in their initial stages; such as:- Uraemia, Post-partum Eclampsia, Acute Yellow Atrophy of the Liver, or Puerperal Septicaemia.

In looking over the previous history of the pregnancy there is nothing suggestive of auto-intoxication beyond occasional attacks of constipation; in fact the patient was severely constipated a fortnight before the onset of labour, but successful measures were taken to rectify this.

There was no persistent vomiting during pregnancy, the ordinary "Morning Sickness" ceasing at about the fifth month.

There was no previous morbid condition of the Liver to be made out, from the history obtained from the patient and from two careful examinations of that organ which were made thus:- the first (three months before pregnancy commenced) giving the upper margin of liver dulness at the fifth rib, the lower margin about two fingersbreadths below the costal margin in the mammary line; the second examination which was made towards the end of the eighth month apparently confirming this.

There was no previous Renal affection, so far as could be ascertained by the condition of the patient, and by the urinary analyses which were made on three occasions during pregnancy, when an average result was obtained as follows:- Specific Gravity 1015: Acid: no Sugar: no Albumin: no Tube Casts.

The Circulatory, Respiratory, and Haemopoietic systems,

shewed no apparent signs of organic disease, by reason of the previous history of the patient's condition, and by examinations made before and during pregnancy.

The patient had not experienced a general anaesthetic before her accouchement.

THE LABOUR.

When the anaesthesia was initiated, she appeared to take the chloroform with difficulty, crying out and resisting in a very excited and "nervous" manner; after this the anaesthetic was apparently well borne.

The amount of chloroform used was approximately five and a half ounces; it was administered on a Schimmelbusch Mask, which allows a free evaporation, and aeration.

The type of anaesthesia induced was intermittent during the pains, and of light intensity; but Surgical Anaesthesia was promoted during (1) the application of Forceps, and the delivery of the child:- for about twenty minutes.

(2) the manual removal of the placenta:- for about five minutes and

(3) during the repair of the Perinaeum:- for about fifteen minutes.

The periods of intermittent anaesthesia give an approximate total of four hours duration; which

includes about forty minutes of Surgical Anaesthesia.

The Labour was conducted with the usual strict antiseptic and aseptic precautions. To emphasise the fact that all available precautions to guard against Sepsis were taken, the following may be mentioned:- In addition to the ordinary technique of labour, all mechanical and manual interference was conducted with gloved hands, two pairs of gloves being in use, which allowed of a sterilised pair being always in readiness: the social position of the patient allowed of a carefully prepared lying-in room, which had been newly painted, and the sanitary conditions of the house (which had been revised some months previously) were all that could be desired.

THE PUERPERIUM.

A recapitulation of the clinical phenomena which were noted is here given, so as to more easily compare the various morbid conditions which had to be tested before the possibly true Diagnosis was reached.

THE FIRST DAY:- The patient had a period of comparative well-being: Pulse 90; Temperature 99° F.

Towards evening she complained of drowsiness and frontal headache; but otherwise she passed a First Day which was typical of any Post Anaesthetic Puerperium.

THE SECOND DAY:- She said "she felt well": but the headache still persisted and there was slight general jaundice, most marked on the face and in the Conjunctivae. The Perineal Wound was healthy, and

the Lochia non offensive. Temperature 99° F. Pulse 100.

Towards night the jaundice was intensified: the headache was acute, she was restless and drowsy, and she complained in addition of a feeling of nausea, thirst, and hiccough. Pulse 116: Temperature 99° F.

It was evident that the patient was seriously ill.

The differential diagnosis of Uraemia, Post-Partum Eclampsia, Acute Yellow Atrophy of the Liver, or Puerperal Septicaemia, in their respective initial stages had to be faced.

A Catheter specimen of urine shewed there to be no Albumin present: this fact made the diagnosis of Uraemia less tenable.

There had been no vomiting, no rigors, and the area of liver dulness was unchanged.

The treatment adopted was expectant, and a guarded prognosis was given.

The supposition that Post Anaesthetic Acid Intoxication might have accounted for the foregoing clinical phenomena had not yet been entertained.

THE THIRD DAY:- The patient had passed a very restless night; the pupils were widely dilated, the jaundice definitely marked, and the smell of acetone present in the breath. Acetone and Diacetic Acid were present in the urine.

In consequence of the above the further possibility of Delayed Chloroform Poisoning was added to the hypothetical diagnosis. The fact that there had been no convulsive seizure, made the supposition of

Eclampsia less likely. Acute Yellow Atrophy of the Liver was still a possibility though there had been no vomiting, and the Liver shewed no signs of atrophy as afforded by percussion.

The strong points in favour of the diagnosis of Yellow Atrophy were the persistent jaundice, the drowsy state, and the Tyrosin-like crystals in the urine.

Puerperal Septicaemia was still dreaded and it was argued that if such a condition was in process at so early a stage, the resulting infection would be due to a very virulent organism, this coupled with the fact of Hepatic inadequacy would have made the prognosis of Septicaemia very grave. Against the diagnosis of Septicaemia were the following points:- (1) The absence of rigors. (2) The free lochial flow, which was not offensive. (3) The healthy condition of the perineal wound and (4) The absence of tenderness over the lower abdomen.

The presence of acetone in the urine and in the breath, determined the provisional diagnosis of Post Anaesthetic Acid Intoxication, and Ant-Acid treatment was exhibited without delay.

THE FOURTH DAY:- Vomiting had started the night before and had persisted with short intermissions: it was not haemorrhagic.

The patient had been delirious during the night; and when seen in the morning responded when spoken to, but could not carry on a rational conversation: Temperature 100.8° F. Pulse 127-140.

The breath was charged with acetone, the skin markedly jaundiced especially on the face and in the conjunctivae.

As she lay in bed, she presented a striking clinical picture:- she lay occasionally tossing restlessly from side to side of the bed, moaning and breathing rapidly: she would greedily take a drink and then vomit it up, apparently without effort.

Ant-Acid treatment was pushed: Sodium Bicarbonate being given in drachm doses, frequently.

The fact that the liver dulness was unchanged and the vomiting not haemorrhagic made the diagnosis of Acute Yellow Atrophy of the Liver not so imminent a possibility.

Throughout the Puerperium the lochial flow was free and not offensive: the temperature was never above 100.8° F.

Further, it should be noted that there was marked improvement by ten p.m. on the same day (the fourth), when the skin was acting freely and the flow of urine increased; and that on the following day all the adverse signs and symptoms had disappeared, the patient ultimately progressing to a complete recovery.

It is not warranted to say that Sepsis was not present, for though it was negatived at the bedside, no bacteriological examination was made to determine whether the Blood was sterile, nor was there a leucocyte count taken.

MacDonald¹⁴ drew attention to a case occurring in the Edinburgh Maternity Hospital, which is not

completely analogous to the writer's case¹⁵, as the patient had one convulsive seizure before delivery:- the patient was safely delivered by forceps under chloroform: two days after she became gravely ill, she was intensely jaundiced: there were no indications of Sepsis, the Blood was sterile, and there was no leucocytosis. She made an uneventful recovery.

Again, Anning¹⁶ published a case in which the patient, a primipara with no antecedent kidney disease, had violent symptoms of poisoning with jaundice after labour in which chloroform was the anaesthetic used. This case differs from the writer's in that the symptoms appeared with greater rapidity, the pulse-rate being more frequent, the delirium more marked, and the vomiting being haemorrhagic, and also occurring on the first day - all differences of degree only. The material difference being that the Liver dulness shewed diminution in size as recorded by percussion and was again almost "normal" in ten days.

The cause assigned was Acute Yellow Atrophy of the Liver.

It is a recognised fact that Delayed Chloroform Poisoning and Acute Yellow Atrophy may produce similar symptoms; but while the latter is a rare disease, symptoms of the former are not, although probably very rare in cases of parturition. Therefore the writer

ventures to suggest that Anning's case is more easily explained by the assumption that the signs and symptoms observed were due to the delayed action of the chloroform, than by the assigned cause of Acute Yellow Atrophy of the Liver.

MacDonald¹⁴ holds that the post-mortem changes following these conditions are separate and distinct: the Liver in Delayed Chloroform Poisoning shewing an intense fatty change, with a necrosis of the central zones of the lobules, but never the total necrosis of the whole lobule so characteristic of Acute Yellow Atrophy: again there is no proliferation of bile ducts, probably due to the acuteness of the process, as is present in the Liver in Acute Yellow Atrophy.

It is admitted that they are both Acid Intoxications; and further that both conditions may follow Chloroform Narcosis: though, of course, typical Acute Yellow Atrophy of the Liver occurs independently of any Anaesthetic.

Hence it is probable that the clinical symptoms (occurring in such cases, after the administration of chloroform, and which so closely resemble those of Acute Yellow Atrophy of the Liver) are caused by the unexplained action of the anaesthetic itself.

The clinical phenomena met with in Case I. determined the writer to adopt prophylactic measures with reference to all future cases of parturition, with a view to subsequent Chloroform Anaesthesia.

The ordinary procedure during pregnancy, as regards the careful regulation of the bowels, was to be strictly enforced whenever necessary; in addition, if digestive disturbances were present during the last months, it was proposed to exhibit Sodium Bicarbonate; and further, it was proposed to regulate the diet, by reducing the intake of Proteids and Fats during the last month, and to proportionately increase the usual amount of Carbohydrates.

It should be here noted that the Yorkshire Farming Class eat immoderately of animal food as a rule.

It was argued that if such a procedure was carried out, the parturient woman would undergo her labour in the best state of health possible to cope with the post-anaesthetic acidosis: if such were proved to be present.

As regards the cases, the following routine procedure was formulated:- a careful history was to be obtained (before labour whenever possible) of any previous confinement, whether chloroform had been administered, and if so, whether there had been any vomiting, headache, or jaundice during the first few

days of the puerperium.

The Liver dulness was to be determined as early in the pregnancy as possible and the results obtained were to be compared with those present during the puerperium.

The ordinary urinary analysis before labour was to be supplemented by the qualitative tests for Acetone; while during the puerperium it was proposed to estimate the daily amount of urine, the quantity of urea excreted, to examine for Acetone and Lactose, and finally to microscopically examine the deposit for tube casts, crystals etc.

In addition the amount of chloroform used, and the approximate duration of the anaesthesia were to be noted.

CASES II. III. AND IV.

During the pregnancy of these three cases, there had been troublesome constipation; and as they were first seen in each case only a few days before labour commenced, the bowels were not strictly regulated nor were the precautions carried out as to diet.

CASE II. gave a history of vomiting after two confinements in which chloroform had been administered, further she said she had been jaundiced during the puerperium on one of these occasions,

In all three cases during the puerperium Acetonuria

was present, they all shewed slight jaundice, which was clearly demonstrable in the conjunctivae, and frontal headache and nausea were experienced.

Further, the liver dulness was apparently unchanged as compared with the previous measurements; and the lochial discharges were free and not offensive.

Case II. vomited twenty four hours after the cessation of labour.

In the puerperium of these Cases, as headache and nausea were present during the first day, Sodium Bicarbonate was administered in large doses with a very free fluid diet, in accordance with the treatment advocated in post anaesthetic cases by Beesly⁸, Langdon Brown⁴ and others.

The "malaise" after the labour was admittedly slight but from the phenomena observed it was concluded that all three patients shewed a slight degree of acid intoxication which was due to the action of the chloroform.

CASE II.

Mrs H. aet 37: Multipara: Farmer's Wife.

She was a large muscular woman, of the brunette type. PREVIOUS HEALTH "Always good" except for troublesome attacks of constipation.

She said that "in two of her five previous confinements she had chloroform administered, both of these were instrumental cases: on both occasions she suffered from severe headache and vomiting for the first two or three days of the puerperia: she recollected the nurse remarking, that she was jaundiced during one puerperium".

PREGNANCY. This had been complicated by severe and obstinate constipation; there had been no excessive vomiting.

The patient was first seen only two days before the onset of labour.

The upper margin of the liver dulness was in the fourth interspace, the lower margin being approximately one fingersbreadth below the costal margin in the mammary line.

There was no disease apparent in the heart and lungs

A specimen of urine gave the following:- Specific Gravity 1022: very Acid: no Albumin, Sugar, nor Acetone: no Tube Casts.

THE LABOUR. When first attended, the second stage was in progress; the presentation was vertex: Right Occipito Posterior.

A living child was duly delivered by forceps: the perinaeum was not lacerated.

An intravaginal douche was given after the termination of labour.

The duration of chloroform anaesthesia extended over a period of approximately two and a half hours: three ounces were administered on a Schimmelbusch mask.

PUERPERIUM.

FIRST DAY. One hour after the termination of labour ten ounces of urine were drawn off by Catheter (See Chart).

When seen later the patient had slept at intervals all day: Temperature 98° F. Pulse 100.

She complained of a frontal headache, and nausea: no jaundice was noted, the tongue was furred; and there was no acetone smell in the breath.

Abundance of fluid nourishment was ordered, with frequent drinks of water, milk and soda etc: Sodium Bicarbonate (sixty grains) was ordered every two hours.

Twenty ounces of urine were passed naturally late at night.

SECOND DAY. The headache had interfered with her sleep: she had vomited twice between four and six a.m. (the vomit was not kept for examination, it was said to have consisted of "green matter", no "coffee ground like" substances were said to have been present).

Temperature 99° F. Pulse 108.

The conjunctivae were tinged pale yellow, and on close inspection the skin over the face, neck, and

chest, was slightly jaundiced: the tongue was coated with a yellow fur: no acetone smell was noted in the breath.

The lochial discharge was free and not offensive.

The liver shewed no signs of alteration in size as evidenced by percussion.

Castor Oil was ordered to be given at night.

THIRD DAY. The bowels had moved freely in the early morning: she had passed a very fair night, her headache had disappeared and there had been no vomiting. Temperature 98.8° F. Pulse 90.

Her face was of a ~~pale~~ yellow ivory colour, not so yellow as the day before, though the conjunctivae still shewed slight signs of jaundice. The tongue was nearly clean.

Lactation was in process, the breasts being engorged with milk.

She had passed urine in much larger amounts. The Sodium Bicarbonate was withheld.

FOURTH DAY. The Jaundice had disappeared.

The further progress was uneventful.

THE URINE - CASE II.

A specimen two days before labour gave:- Specific Gravity 1022: Very Acid: No Albumin, Acetone, nor Sugar: the deposit shewed Urates, but no tube Casts were found.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone	Lactose	Deposit	U.	A.
1	1029	Very Acid	Nil.	Nil.	B.C. Urates Uric Acid.	15 450	10 20
2	1028	Very Acid	Present.No Diacetic Acid.	Nil	ditto	10 300	30
3	1026	Acid	Present	Present	B.C. and Urates only	8 408	51
4	1022	Neutral	Nil.	Nil.	ditto	10	?
5	1020	Slight Acid.	Nil.	Nil.	A few B.C.	8 328	41

The Catheter specimen on the first day contained Albumin, no Acetone was noted: the Reaction was

markedly Acid, Urates and Uric Acid being present in the deposit.

In the Chart under 'U' the top figure indicates the amount of urea in grains per ounce, the lower, the total amount of urea excreted.

The colour given by the foam in the Sodium Nitroprussiate Test was a moderate purple on both days on which acetone was present.

It will be seen that no diacetic acid was found: lactose was present on the third day.

CASE III.

Mrs S. aet 36: multipara: Farmer's Wife.

PREVIOUS HISTORY. A strongly made dark haired woman: who said "she had always perfect health".

In all her previous six pregnancies there had been troublesome constipation: though internal version and instruments had been employed in two of her previous confinements, no chloroform had ever been administered.

The patient gave the history of puerperal fever six years ago.

PREGNANCY. The patient was first seen on the day before labour set in: there had been very obstinate constipation.

The upper margin of liver dulness extended from the fifth rib to the costal margin in the mammary line.

The urine:- Specific Gravity 1026: very Acid: no Albumin, Acetone, nor Sugar: in the deposit no Casts were observed but abundant urates were present.

LABOUR. Was simple

One and a half ounces of chloroform were administered on a Schimmelbusch mask for a period of approximately one hour and a half.

PUERPERIUM.

FIRST DAY. Temperature 98.6° F. Pulse 90.

She complained of frontal headache, and nausea: she took nourishment well. Sodium Bicarbonate (sixty grains) was ordered every two hours.

SECOND DAY. She was slightly jaundiced, round the eyes, and in the conjunctivæ: the tongue was furred; she had no headache; and there had been no vomiting.

Temperature 98.4° F. Pulse 97. Castor Oil was ordered at night.

THIRD DAY. Temperature 99.2° F. Pulse 98.

Lactation had set in: there was no nausea, headache nor vomiting.

Slight jaundice was still apparent and the tongue was almost clean.

The bowels had moved freely: the Sodium Bicarbonate was withheld.

FOURTH DAY. The conjunctivae still shewed slight jaundice which was not apparent on the face: she was very well.

Her further progress was uneventful, the conjunctivae being quite clear of jaundice on the next day.

URINE.

A specimen of urine the day before labour gave:-
Specific Gravity 1026: very Acid: no Albumin, Acetone, nor Sugar: in the deposit no Casts were observed but abundant urates were present.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone.	Lactose	Deposit	U.	A.
1	1026	Acid very	Nil.	Nil.	B.C. Uric & Urates.	14 420	30
2	1029	Acid very	Present.	Nil.	No Uric abundant Urates.	12 372	31
3	1026	Slight Acid	Present.	Nil.	ditto.	8 384	48 ?
4	1022	Acid	Nil.	Nil.	ditto.	8 288	36 ?
5	1019	Slight Acid	Nil.	Nil.	-	7 280	40

The colour obtained by the Sodium Nitroprussiate Test was a moderate purple, on both days: the colour being deeper on the second day.

The test for Diacetic acid was negative. The deposit of urates on the second was markedly heavy.

CASE IV.

Mrs P. aet 27: Primipara: Farmer's Wife.

She was a well developed woman, dark haired and of medium height.

PREVIOUS HEALTH. "Always good".

PREGNANCY. She was first seen fourteen days before the onset of labour: there had been no undue vomiting but she had suffered greatly from constipation during the last month.

As far as could be ascertained the liver dulness extended from the fourth interspace to the costal margin in the mammary line.

LABOUR. Simple: it was very tedious owing to a large child (nine lbs): the perinaeum was not torn. Four ounces of chloroform were used, being given on a folded towel: the period of intermittent anaesthesia extended over approximately four hours.

PUERPERIUM.

FIRST DAY. She was very tired: complaining of frontal headache: she wanted to vomit but did not. Temperature 99° F. Pulse 104. Sodium Bicarbonate was ordered sixty grains every two hours.

SECOND DAY. She had slept at intervals but had been rather restless during the night. The nausea and headache had passed off.

Pulse 88. Temperature 99° F.

Her tongue was slightly furred: the conjunctivae were slightly jaundiced. She "felt very well". Castor Oil was ordered at night and the Sodium Bicarbonate mixture withheld.

THIRD DAY. She was much improved. Lactation was in process: the bowels moved well later in the day.

Further progress was uneventful.

URINE.

A specimen twelve days before labour gave:- Specific Gravity 1023: Acid: no Albumin, Acetone, nor Sugar, no Casts were noted in the deposit, but some epithelial cells, probably of vaginal origin, were noted.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone.	Lactose	Deposit	U.	A.
1	1030	Acid very	Present	Nil.	B.C. Uric scanty. Urates.	12' 432	36
2	1028	Acid very	Present	Nil.	B.C. and Urates.	10 390	39

CHART (continued)

P.	S.G.	R.	Acetone.	Lactose	Deposit.	U.	A.
3	1026	Acid	Present	Present	B.C. and Urates	8 440	55
4	1020	Slight Acid	Nil.	Nil.	Phosphates and Urates	6 300	50
5	1018	Slight Acid	Nil.	Nil.	-	6 288	48

Note: that Acetone was present on the first three days no diacetic acid was found.

The colour in the test for acetone was a deep purple foam on the first and second days: the colour very less marked on the third day.

CASE V.

The following case gave a previous history of slight jaundice, with occasional attacks of pain and vomiting, probably due to some Hepatic obstruction, possibly Gall Stones.

Acetonuria was present during the ninth lunar month of pregnancy as will be seen by the Chart.

Chloroform was well borne during the labour.

Slight headache and nausea were complained of during the first day of the puerperium and acetone was present in the puerperal urine for the first four days.

Mrs X. aet 34: Primipara. She was a large "flabby" woman, dark haired and "neurotic": on casually looking at her face, the sallow complexion and "muddled" conjunctivæ suggested Jaundice.

PREVIOUS HISTORY. She had complained of occasional sharp attacks of pain over the upper region of the abdomen on the right side during the last ten years: these attacks were intermittent, at times not occurring for months, and were usually accompanied by a moderate degree of jaundice: she said she had never passed a gall stone so far as she knew.

There had been chronic constipation: she had used

an enema regularly till two years ago, when dieting and the administration of Nux Vomica, and Aloes had rendered that unnecessary.

No Albumin, nor bile was found in the urine of the last two years.

There was tenderness over the region of the gall bladder on one occasion when the abdomen was examined.

An exploratory laparotomy had been suggested, and refused.

The heart and lungs, so far as repeated examinations shewed, were healthy.

PREGNANCY. At the first month, the liver dulness extended from the fourth interspace to two fingers-breadths below the costal margin in the mammary line; giving an approximate vertical diameter of six and a half inches.

There was no undue vomiting throughout the pregnancy; and there were no "liver attacks", with definite jaundice.

Rigid precautions were enforced to provide a free action of the bowels:- Castor Oil, or equal parts of Sulphur and Cream of Tartar (a.a. half a drachm) were used for alternate weeks during the last three months, whenever any indication of their use was indicated.

In addition Sodium Bicarbonate (grains 15 thrice daily) was prescribed for the same period, during alternate weeks.

For the urine during pregnancy see Chart.

THE LABOUR: was simple: a large healthy child was eventually delivered.

The perinaeum was not torn.

Two ounces of chloroform were used, the period of intermittent anaesthesia being approximately of three hours duration.

PUERPERIUM.

FIRST DAY. She was very tired, complaining of nausea and slight headache. There was no vomiting.

Temperature 99° F. Pulse 90.

Sodium Bicarbonate (half a drachm) was exhibited every two hours.

The conjunctivæ shewed slight jaundice.

SECOND DAY. The headache and nausea had passed off.

Her complexion was, if anything, slightly yellower than usual, the conjunctivæ still shewing slight jaundice; her complexion frequently shewed a yellow

tinge even at ordinary times).

Temperature 98.8° F. Pulse 92.

Lactation was in process.

Castor Oil was administered at night.

THIRD DAY. She had slept well; the bowels moved satisfactorily after a copious enema of soap and water.

Temperature 99° F. Pulse 88.

The liver dulness was unchanged; there was slight tenderness on palpation over the region of the gall bladder.

The conjunctivae shewed their usual "muddied" appearance; jaundice could not be said to be present.

The Sodium Bicarbonate was withheld early in the day.

FOURTH DAY. She was very well and progressed to an uneventful recovery.

THE URINE OF CASE V.

Chart of Urine during Pregnancy.

Month Lunar	S.G.	R.	Albumin	Acetone	Sugar	Deposit
8th	1023	Acid	Nil.	Nil.	Nil.	Urates No Casts
9th	1025	Acid	Nil.	Present	Nil.	Urates No Casts
"	1023	Acid	Nil.	Present	Nil.	Urates
"	1020	Acid	Nil.	Nil.	Nil.	Slight Urates.
"	1019	Slight Acid	Nil.	Nil.	Nil.	Nil.
10th	1022	Acid	Nil.	Nil.	Nil.	Slight Urates.

Though Acetone was present during the ninth lunar month, there were no toxic symptoms present.

CHART OF URINE DURING PUERPERIUM.

P	S.G.	R.	Acetone	Lactose	Deposit	U.	A.
		very			Dense Urates	15	
1	1030	Acid	Present	Nil.	Uric Acid.	540	36
					Urates	8	
2	1026	Acid	Present	Nil.	No Uric.	360	45
					Slight	7	56
3	1024	Acid	Present	Nil.	Urates	392	?
			(?)		Slight	6	
4	1014	Neutral	Present	Nil.	Urates	360	60
						7	
5	1017	Acid	Nil.	Nil.	-	336	48

No diacetic acid was found. The reaction for Acetone was doubtful on the fourth day: the foam in the acetone reaction was of a deep purple on the first three days, the colour being most marked on the second day: on the fourth day the colour was very faint.

The amount of urea excreted on the first day was tested three times; as the amount was so large it was thought that there had been an error in the technique (Ureometer of Doremus).

CASES VI. VII. VIII. IX. X. XI. XII. XIII. XIV.

These Cases have the following points in common:-

During the Pregnancy there was no obstinate constipation, nor was there any undue vomiting, (except in Case IX. in which occasional undue attacks of dyspepsia and vomiting occurred).

Prophylactic measures were taken in each case, by guarding against Constipation, and by restricting the diet with regard to proteid and fat (during the last month): the amount of Carbohydrate being increased. In Case IX. Sodium Bicarbonate was administered during the later months of pregnancy by reason of the Dyspeptic symptoms noted.

The Labour in each Case was simple; and intermittent Chloroform anaesthesia of slight degree was induced.

There was no definite "malaise" during the early days of the Puerperium, e.g. no headache, nausea, nor jaundice: accordingly Sodium Bicarbonate was not administered except in the first three days of the Puerperium of Case IX. when it was exhibited by reason

of the dyspeptic symptoms present.

Acetonuria was present in each case, during the puerperium.

It is interesting to note that Case XI. gives a previous history of headache and nausea occurring early in the puerperium of a confinement in which chloroform had been administered and forceps applied.

CASE VI.

Mrs D. aet 33: multipara.

She was a strong healthy woman.

PREVIOUS HEALTH. "Was perfect" for as long as she could remember.

In her previous confinement (her first) chloroform had been administered: she had no untoward signs or symptoms during the puerperium such as could be attributed to the action of the anaesthetic.

There was no apparent affection of the heart or lungs.

PREGNANCY. Uneventful.

The upper margin of liver dulness was at the fifth rib, the lower margin being apparently one fingers-breadth below the costal margin in the mammary line.

URINE. See Chart.

LABOUR. Simple: the Child was just born on arrival.

As the placenta was adherent, chloroform was administered, two drachms being used on a towel for about ten minutes.

PUERPERIUM.

FIRST DAY. She was tired: there was no headache nor nausea.

Temperature 99° F. Pulse 90.

SECOND DAY. She had passed a good night, no jaundice was apparent, the lochial discharges were free and not offensive.

The liver dulness was unchanged.
Temperature 98.2° F. Pulse 80.

THIRD DAY. Lactation was in process.
Temperature 99° F. Pulse 86.
Further progress was uneventful.

THE URINE.

A specimen of urine during pregnancy gave the following:- Specific Gravity 1020: Acid: no Albumin, Acetone, Sugar, nor tube Casts.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone.	Lactose	Deposit	U.	A.
1	1027	Acid	Present	Nil	B.C. Abundant Urates.	12 420	35
2	1023	Acid	Present	Nil	B.C. Urates.	10 380	38
3	1024	Acid	Nil.	Nil.	Slight Urates	9 360	40
4	1025	Slight Acid	Nil.	Nil.	B.C. Scanty	10 440	44
5	1020	Acid	Nil.	Nil.	-	7 266	38

Acetone was present on both days by Lieben's test only (See tests for Acetone page 59-60).

CASE VII.

Mrs C. aet 27: primipara.
An "anaemic looking" woman.

PREVIOUS HEALTH. She said she had been anaemic as a girl; and that she was very liable to constipation.
No apparent disease of any organ was found on examination.

PREGNANCY. Uneventful. No undue vomiting; any apparent constipation yielding to treatment.

Liver dulness:- upper margin at the fifth rib, the lower approximately two fingersbreadths below the costal margin in the mammary line. For urine see Chart.

LABOUR. Was simple: chloroform was administered intermittently for a period extending over two hours. One ounce was used.

PUERPERIUM.

FIRST DAY. Late on in the day she was very thirsty, and complained of the taste of chloroform in her mouth; otherwise she was very well, taking nourishment freely. Temperature 98.6° F. Pulse 89.

SECOND DAY. In excellent health. Castor Oil was ordered.

THIRD DAY. Temperature 99.4° F. Pulse 89. Lactation was in process. The Bowels moved freely. The liver dulness was unchanged. Further progress was uneventful.

THE URINE.

A specimen of urine during pregnancy gave the following result:- Specific Gravity 1014: Acid: no Albumin, Acetone, nor Sugar: in the deposit no tube Casts were found, but a few epithelial cells which were probably from the vaginal tract.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone.	Lactose	Deposit.	U.	A.
1	1029	Acid	Present.	Nil.	B.C. Abundant Urates.	14 560	40
2	1022	Acid	Present	Nil.	B.C. Slight Urates.	8 400	50 ?
3	1026	Acid	Nil.	Present	B.C. Mucus & Urates.	10 420	42
4	1019	Acid	Nil.	Nil.	-	6 306	51
5	1020	Acid	Nil.	Nil.	-	7	?

The colour reaction by Legal's Sodium Nitroprussiate Test was evidenced by a moderate purple foam on both days.

CASE VIII.

Mrs A. aet: 41. Multipara.

PREVIOUS HEALTH. She has always been a strong and healthy woman.

She apparently had never any complicated labour, nor had she ever experienced Chloroform anaesthesia during any of her six previous confinements.

PREGNANCY. Uneventful: no undue vomiting, no definite constipation.

The liver dulness:- upper margin was at the fourth rib, the lower being at the costal margin in the mammary line.

THE LABOUR: was simple; chloroform anaesthesia being administered for about twenty five minutes on an open mask: four drachms being used.

PUERPERIUM.

FIRST DAY. Temperature 99.2° F. Pulse 80.

The patient was rather tired; no headache nor nausea was experienced.

SECOND DAY. She had passed a good night, and was very well. Lactation was established on the third day when the Temperature was 98.8° F. The liver dulness was unchanged. The further progress was uneventful.

THE URINE.

A specimen of urine during pregnancy gave the following:- Specific Gravity 1019: Acid: no Albumin, Acetone, Sugar, nor tube Casts.

CHART OF URINE DURING PUERPERIUM.

P	S.G.	R.	Acetone	Lactose	Deposit	U.	A.
1	1026	Very Acid	Present	Nil.	B.C. Slight Urates Uric Acid.	14 546	39
2	1024	Acid	Present	Nil.	B.C. Slight Urates.	12 456	38
3	1026	Acid	Present	Nil.	B.C. Slight Urates.	12 360	30 ?
4	1020	Acid	Nil.	Nil.	-	6 216	36 ?
5	1019	Slight Acid	Nil.	Nil.	-	7 273	39 ?

The Acetone reaction was a deep purple colour on the second day; on the third day the colour was of a horse chesnut hue.

CASE IX.

Mrs R. aet. 27. Primipara.

PREVIOUS HISTORY. "Always healthy".

PREGNANCY. There was troublesome vomiting and gastric discomfort during the second third and fourth months; after which she vomited occasionally till full term.

There was no definite constipation.

The liver dulness, at the third month:- upper border at the fifth rib; the lower being one fingers breadth below the costal margin in the mammary line. (In consequence of the dyspeptic symptoms Sodium Bicarbonate (fifteen grains) was administered thrice daily throughout the pregnancy,) *when necessary.*

LABOUR. Simple: a large healthy child (9 lbs) was delivered. The perinaeum was slightly torn.

Chloroform was administered, for a period extending over five hours: four ounces being used.

PUERPERIUM.

FIRST DAY. Temperature 99.2° F. Pulse 84.

She was very tired, and slept most of the day. Although no nausea, vomiting, headache, nor jaundice were noted, she was ordered Sodium Bicarbonate (grains 40 every four hours) in consequence of the gastric disturbance during pregnancy.

The urine was drawn off by catheter (See Chart)

SECOND DAY. Temperature 98.4° F. Pulse 80.

Patient was very well indeed.

Castor Oil ordered at night.

THIRD DAY. Temperature 98.8° F. Pulse 78: the Bowels moved freely.

Lactation was in process.

Sodium Bicarbonate withheld.

FOURTH DAY. Temperature 99° F. Pulse 100.

The breasts were very turgid: she complained of headache and slight nausea, apparently due to the process of lactation.

There was no jaundice.

The liver dulness was unchanged.

FIFTH DAY. She was very well: the excessive turgescence of the breasts had begun to subside.

Further progress was uneventful.

THE URINE.

During pregnancy the urine was examined during the third, fourth, fifth and ninth months, when an average analysis was obtained as follows:- Specific Gravity 1021: Acid: no Albumin, Acetone, nor Sugar: the deposit contained Urates; no tube Casts were found.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone.	Lactose	Deposit.	U.	A.
1	1026	Very Acid.	Present	Nil.	B.C. Dense Urates. Uric Acid(slight)	13 559	20 23 c
2	1022	Acid	Present	Nil.	B.C. Urates.	10 280	28 ?
3	1026	Slight Acid	Nil.	Nil.	B.C. Urates.	9 378	42
4	1028	Neutral	Nil.	Present	B.C. Urates	12 312	26 ?
5	1023	Slight Acid	Nil.	Nil.	B.C. Slight Urates.	10 480	48

The colour of the foam in the reaction for acetone was a moderate purple, which had faded on the second day.

In the catheter specimen on the first day (which yielded twenty ounces) Albumin was present; but no Casts were found.

Diacetic Acid was not found. As will be seen Lactose was present on the fourth day.

CASE X.

Mrs H. L. aet: 38. Multipara.

PREVIOUS HEALTH. She said "she had always been exceptionally healthy".

She had experienced five previous confinements, chloroform was administered in two of them: so far as she could remember there was no headache nor nausea during the respective puerperia.

PREGNANCY. No undue vomiting nor troublesome constipation were present.

The liver dulness gave the upper margin in the fourth interspace, the lower being at the costal margin in the mammary line.

THE LABOUR. Was simple: a living child was delivered: one and a half ounces of chloroform were used for a period extending over two hours.

The puerperium was uneventful, no nausea, headache, nor jaundice were observed.

The liver dulness was unchanged.

THE URINE.

A specimen of urine during pregnancy gave the following:- Specific Gravity 1021: Acid: No Albumin, Acetone, Sugar, nor tube Casts.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone	Lactose	Deposit	U.	A.
1	1026	Very Acid	Nil.	Nil.	B.C.Urates Slight Uric	12 528	44
2	1022	Acid	Present	Nil.	B.C. Urates.	10 410	41
3	1024	Acid	Nil.	Nil.	B.C. Urates.	9 396	44?
4	1020	Slight Acid	Nil.	Nil.	B.C. Mucus.	8 320	40
5	1021	Acid	Nil.	Nil.	-	8 360	45

No diacetic acid was present.

The colour obtained in performing Legal's test was a faint purple, with the foam of a corresponding colour.

Note that a few uric acid crystals were obtained in the deposit of the first day.

CASE XI.

Mrs W. aet; 44. Multipara.

PREVIOUS HEALTH. Always good.

There had been six previous confinements: chloroform had been administered in four of them; she can remember feeling very sick, and suffering from a severe headache after one confinement in which chloroform had been used.

THE PREGNANCY. There was no undue vomiting: she suffered from slight constipation, which yielded to appropriate treatment.

The upper margin of liver dulness was at the fifth rib: the lower being one fingersbreadth below the costal margin in the mammary line.

THE LABOUR: was simple.

Two drachms of chloroform were used for a period of about twenty minutes.

THE PUERPERIUM.

FIRST DAY. Temperature 99.4° F. Pulse 86.

No nausea, headache, nor jaundice were noted.

SECOND DAY. Temperature 98.4° F. Pulse 80.

Castor Oil was given at night: lactation was in process.

Further progress was uneventful.

The liver dulness was unchanged.

THE URINE.

A specimen of urine during pregnancy gave the following:- Specific Gravity 1016: Acid: No Albumin, Acetone, Sugar, nor tube Casts.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone	Lactose	Deposit	U.	A.
1	1026	Very Acid	Present	Nil.	B.C. Slight Urates	12 504	42
2	1020	Acid	Nil.	Nil.	Phosphates and Slight Urates.	6 324	54?
3	1026	Acid	Nil.	Present	B.C. Slight Urates.	10 400	40?
4	1024	Acid	Nil.	Present	B.C. Slight Urates.	9 342	38
5	1020	Slight Acid	Nil.	Nil.	-	8 264	33

No diacetic acid was present.

The colour obtained in performing Legal's Test was of a faint purple.

Lactose was present on the third and fourth days.

CASE XII.

Mrs L. E. aet: 26. Primipara.

PREVIOUS HEALTH. Always good.

THE PREGNANCY. There was no undue vomiting: no constipation was complained of at all.

The upper margin of liver dulness was in the fourth interspace, the lower at the costal margin in the mammary line.

THE LABOUR. Was simple: there was some delay owing to a very large child, the head being on the perinaeum for about an hour and a half.

The perinaeum was torn, back to but not involving the Anus.

A complete repair was performed at the termination of labour.

Four ounces of chloroform were used being administered over a period of two and three quarter hours.

THE PUERPERIUM.

There was no jaundice, headache, nor nausea present.

The perinaeum healed by first intention.

The liver dulness was unchanged.

Further progress was uneventful.

THE URINE.

A specimen of urine during pregnancy gave the following:- Specific Gravity 1021: Acid: no Albumin, Acetone, Sugar, nor tube Casts.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone	Lactose	Deposit	U.	A.
1	1030	Very Acid	Nil.	Nil.	B.C. Urates & Uric Acid	13 442	34
2	1025	Acid	Present	Nil.	B.C. Urates	10 350	35
3	1024	Acid	Present	Nil.	B.C. -	9 360	40
4	1026	Acid	Nil.	Present	B.C. -	10 380	38
5	1024	Acid	Nil.	Nil.	B.C. -	7 287	41?

No diacetic acid was present.

Legal's test was negative: Lieben's test giving a positive result on the second and third days.

CASE XIII.

Mrs H. aet: 38. Multipara.

PREVIOUS HEALTH. Good.

She had experienced five previous confinements; she had been given chloroform in one of them, with no after effects.

THE PREGNANCY was uneventful.

Liver dulness upper margin in the fourth interspace, lower at costal margin in the mammary line.

THE LABOUR. Was simple. Two drachms of chloroform being used over a period of twenty five minutes.

THE PUERPERIUM.

No Vomiting, jaundice, nor headache were noted. The liver dulness was unchanged. Further progress was uneventful.

THE URINE.

A specimen of urine during pregnancy gave the following:- Specific Gravity 1024: Acid: No Albumin, Acetone, Sugar, nor tube Casts: Urates were present in the deposit.

CHART OF URINE DURING PUERPERIUM.

P	S.G.	R.	Acetone	Lactose	Deposit	U.	A.
1	1028	Acid	Nil.	Nil.	B.C. Urates and Uric.	15 525	35
2	1024	Acid	Present	Nil.	B.C. Urates	8 304	38
3	1025	Acid	Present	Nil.	B.C. -	9 405	45
4	1024	Acid	Nil.	Nil.	B.C. -	8 392	49
5	1018	Acid	Nil.	Nil.	-	7 385	55

No diacetic acid was present.

Legal's test was positive, the resulting colour being of a medium purple, being less marked on the third day.

CASE XIV.

Mrs L. aet: 32. Primipara.

PREVIOUS HEALTH. Always good. She was an exceptionally large woman being five feet eleven inches in height.

PREGNANCY was remarkable, for the excellent state of

health maintained; she never vomited, and there was no constipation.

The upper margin of liver dulness was in the fourth interspace, the lower just below the costal margin in the mammary line.

THE LABOUR. Was simple, but tedious. A large living child was delivered under chloroform: of which four ounces were used over a period of three and a half hours.

The puerperium was uneventful; no headache, nausea, nor jaundice being observed.

On the third day she experienced headache and nausea: as her breasts were very turgid this was evidently caused by lactation.

The liver dulness was unchanged.

Further progress was uneventful.

THE URINE.

A specimen of urine during pregnancy gave the following:- Specific Gravity 1018: Acid: No Albumin, Acetone, Sugar, nor tube Casts.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone	Lactose	Deposit	U.	A.
1	1030	Very Acid	Present	Nil.	B.C. Slight Urates.	12 528	44
2	1025	Acid	Present	Nil.	B.C. -	12 420	35
3	1026	Acid	Present	Present	-	10 400	40?
4	1024	Acid	Nil.	Nil.	-	7 336	48
5	1024	Acid	Nil.	Nil.	-	7 343	49

No diacetic acid was present.

It should be noted that Acetone was present on the first three days: on the first day the colour obtained in performing Legal's test was a deep purple; which was less marked on the two following days, on the last day being of a horse chesnut colour.

FIVE CASES OF LABOUR IN WHICH

THERE WAS NO CHLOROFORM.

The following five Cases experienced uneventful pregnancies and simple labour.

In each case the patient was a strong healthy woman, with no previous history of any antecedent disease, of the Circulatory, Respiratory, Renal, or Digestive Systems.

There was no Acetone obtained in the urine during the puerperium of any of the cases.

THE URINE OF CASE XV.

A specimen of urine obtained during pregnancy, gave the following:- Specific Gravity 1017: Acid: No Albumin, Acetone, Sugar, nor Casts.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone	Lactose	Deposit	U.	A.
1	1026	Very Acid	Nil.	Nil.	B.C. Slight Urates.	12 480	40
2	1026	Acid	Nil.	Nil.	B.C. Slight Urates.	9 324	36
3	1029	Acid	Nil.	Present	B.C. Urates	8	?
4	1027	Acid	Nil.	Present	B.C. -	7 371	53
5	1020	Acid	Nil.	Nil.	-	8 376	47

THE URINE OF CASE XVI.

A specimen of urine obtained during pregnancy, gave the following result:- Specific Gravity 1020: Acid: No Albumin, Acetone, Sugar, nor Casts.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone	Lactose	Deposit	U.	A.
1	1027	Acid	Nil.	Nil.	B.C. Urates	14 490	35
2	1026	Acid	Nil.	Nil.	B.C. Slight Urates	12 384	32
3	1024	Acid	Nil.	Nil.	-	9 432	48
4	1025	Acid	Nil.	Present	-	8 392	49
5	1024	Acid	Nil.	Nil.	-	8 360	45

THE URINE OF CASE XVII.

A specimen of urine obtained during pregnancy, gave the following result:- Specific Gravity 1021: Acid: No Albumin, Acetone, Sugar, nor Casts.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone	Lactose	Deposit	U.	A.
1	1027	Acid	Nil.	Nil.	B.C. Urates & Uric Acid	12 468	39
2	1020	Acid	Nil.	Nil.	B.C. Urates	6 270	45?
3	1025	Acid	Nil.	Nil.	B.C. -	9 378	42
4	1024	Acid	Nil.	Nil.	-	6 348	58
5	1020	Acid	Nil.	Nil.		8 400	50?

THE URINE OF CASE XVIII.

A specimen of urine obtained during pregnancy, gave the following result:- Specific Gravity 1019: Acid: No Albumin, Acetone, Sugar, nor Casts.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone	Lactose	Deposit.	U.	A.
1	1029	Acid	Nil.	Nil.	B.C. Urates Uric.	11 440	40
2	1024	Acid	Nil.	Nil.	B.C. Slight Urates.	7 308	44
3	1027	Acid	Nil.	Present	-	8 432	54
4	1022	Acid	Nil.	Nil.	-	8 400	50
5	1020	Acid	Nil.	Nil.	-	7 364	52

THE URINE OF CASE XIX.

A specimen of urine obtained during pregnancy gave the following result:- Specific Gravity 1023: Acid: No Albumin, Acetone, Sugar, nor Casts.

CHART OF URINE DURING PUERPERIUM.

P.	S.G.	R.	Acetone	Lactose	Deposit.	U.	A.
1	1030	Very Acid	Nil.	Nil.	B.C. Urates	10 460	46
2	1026	Acid	Nil.	Nil.	-	8 336	42
3	1028	Acid	Nil.	Nil.	-	9 405	45?
4	1023	Acid	Nil.	Nil.	-	7 364	52
5	1024	Acid	Nil.	Nil.	-	8 408	51?

DISCUSSION OF THE URINE, OF THE NINETEEN CASES
EXAMINED, BEFORE AND AFTER LABOUR.

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The following conditions of the urine during pregnancy should be mentioned.

In no Case was there Albuminuria, as afforded by the tests employed:- Boiling, and Heller's, Nitric Acid Test.

Acetonuria was only demonstrated in Case V. during the ninth lunar month: in this case there was the previous history of occasional attacks of pain over the region of the gall bladder with jaundice, of about ten years duration.

When acetonuria was present in Case V. there were no concomitant toxic symptoms present, e.g. jaundice, headache, nausea, or vomiting.

No Sugar was present in any case as evidenced by Fehling's test.

In the examination of the deposit of the urines of these cases, tube casts were never demonstrated: but occasionally epithelial cells were noted, especially during the approach of full term; these cells were probably of vaginal origin.

Urates were noted in varying amount: Uric Acid was never demonstrated.

The opportunity is here taken of stating that the appended analyses of the amounts of urine excreted are not definitely accurate in all cases, by reason of the difficulties experienced in the collection of the urine which were chiefly due to the carelessness of the attendant.

THE URINE WITH REFERENCE TO THE PUERPERAL STATE.

In the fourteen post anaesthetic cases, the average daily amount of urine excreted during the first five days of the puerperium was as follows:-

36.5	ounces	were	excreted	on	the	first	day
36.8	"	"	"	"	"	second	day
43	"	"	"	"	"	third	day
43.6	"	"	"	"	"	fourth	day
48	"	"	"	"	"	fifth	day

As the excretion of Urea was not estimated in Case I, the average amount is calculated on the urine of thirteen cases only.

(The average amount is here given in grains per ounce, multiplied by the average daily quantity of urine, and the resulting average daily amount in grains)

Urea.	13.2	x	Urine	37.5	=	495	grains	on	the	first	day
"	9.2	x	"	38.6	=	355.1	"	"	"	second	"
"	9.6	x	"	43.7	=	419.5	"	"	"	third	"
"	8.1	x	"	42.6	=	345	"	"	"	fourth	"
"	7.3	x	"	43.6	=	318.2	"	"	"	fifth	"

As there were apparent toxic symptoms in Cases I. II. III. and IV., a separate average is worked out for the amount of urine and urea during the first five days.

The urea average is necessarily calculated on the urine of Cases II. III. and IV.

The amounts of urine are here given for cases I. II. III. and IV.

30.2	ounces	of	urine	excreted	on	the	first	day
28.5	"	"	"	"	"	"	"	second day
46	"	"	"	"	"	"	"	third day
39	"	"	"	"	"	"	"	fourth day
57.2	"	"	"	"	"	"	"	fifth day

The average quantity of urea for Cases II. III. and IV. is as follows:- the urea in grains per ounce, multiplied by the average daily amount of urine (for the three cases), and the resulting average daily amount in grains.

Urea	13.6	x	Urine	32	=	435.2	grains	on	the	first	day.
"	10.6	x	"	33.3	=	352.9	"	"	"	second	day.
"	8	x	"	51.3	=	410	"	"	"	third	day.
"	8	x	"	41.6	=	332.8	"	"	"	fourth	day.
"	7	x	"	43	=	301	"	"	"	fifth	day.

Hence according to the results obtained the average daily amount of urine excreted during the first day of the puerperium is 36.5 ounces, which is less than the

average amount passed during ordinary conditions of health, which is generally stated to be from 45 to 50 ounces.

If the average daily quantity be considered for the first four cases only, when certain varying degrees of toxæmia were present, it will be seen that the result is still lower, there being thirty ounces on the first day, and even less on the second when the average is 28.5 ounces. (The low reading on the second day is influenced by the small amount passed by Case I. when only fourteen ounces were registered.)

As regards the excretion of urea:-

The average amount excreted per diem is about 450 grains in ordinary conditions of health.

In the thirteen cases here given, the average daily amount of urea during the first day of the puerperium is 495 grains; this falls on the second day to 355, while on the third there is a rise to 419 grains, after which the average amount again falls.

Hence there is an increase in the output of uræa on the first day as evidenced by the average of the urine of thirteen cases, which shewed no apparent renal condition previous to the labour.

The increase on the first day may be expected, by reason of the increased muscular work incident to the

labour: and further, the decrease in the amount of the second day may be explained, by assuming that the tissues were in a "state of reaction" after the previous extra elimination of the metabolic products caused by the increased muscular strain of parturition. The subsequent rise of the urea on the third day, may be explained by the processes incident to lactation.

Lactosé was present in the urine in seven of the thirteen cases, occurring on the third day in five, on the fourth in two of the cases.

And further, it should be noted that the average of the Specific Gravity recorded on the first, second, and third days respectively is 1027, 1024, and 1025.

In the five cases of labour "without chloroform" the average output of urine and urea, is as follows:-

Urea	11.8	grains	x	Urine	40	oz	=	472	grains	on	1st	day
"	8.4	"	x	"	39.8	"	=	334.3	"	"	2nd	"
"	8.6	"	x	"	47.2	"	=	405.9	"	"	3rd	"
"	7.2	"	x	"	52.4	"	=	377.2	"	"	4th	"
"	7.8	"	x	"	49	"	=	382.2	"	"	5th	"

Lactose was present in the urine in three of the five cases, occurring on the third day in Case XVIII., on the third and fourth days in Case XV., and on the fourth day only in Case XVI.

Accordingly Lactose was present in the urine of ten

of the eighteen cases systematically examined, or in 55.5 per cent.

Ney¹⁷ observed Lactose in 77 per cent of his cases, while McCann and Turner¹⁸ detected it in every case examined: on the other hand Williams¹⁹ only found Lactose present in 2.6 per cent of his cases.

As compared with the post anaesthetic cases, the average daily amount of urine (in the non-chloroform cases) is greater throughout the first five days of the puerperium: while the average total output of urea is less, for the first three days; though the average amounts on the fourth and fifth days are 377 and 382 grains, as compared with 345 and 318 grains in the post anaesthetic cases.

The Specific Gravity index is very similar to that in the post anaesthetic cases, being 1027, 1024, and 1026 on the first three days respectively.

The number of cases examined is small being only nineteen, but the fact is apparent that there is a smaller average amount of urine excreted during the puerperium by the fourteen post anaesthetic cases, than there is by the five cases without chloroform.

Further, the average daily output of urea by the thirteen post anaesthetic cases examined is absolutely greater for the first three days of the puerperium,

than the average amount excreted by the five cases without chloroform.

Therefore it is apparent that the results obtained by the foregoing chloroform cases do not altogether agree with the view held by Williams¹⁹, who states that the amount of urine is increased during the first few days of the puerperium, and that the excretion of urea is less than usual.

Albumin was found in the catheter specimens in Cases II. and IX. on the first day; no Casts were noted.

In the urine obtained by catheter just after labour, Williams noted the presence of Albumin and Hyaline casts, the former in 62.9 per cent, the latter in 19.46 per cent of 1,000 Cases examined.

Further, he holds that the presence of the Albumin and Casts is transient, apparently resulting from the general systemic strain associated with labour, and has no prognostic significance. Temesvary²⁰ has recorded results similar to Williams.

ACETONE.

Acetone was not found to be present by the colour tests in the puerperal urine of any of the five cases of labour without chloroform.

On the other hand acetonuria was demonstrated in all the post anaesthetic cases.

Diacetic acid was present in Case I. on one occasion only, it was not found in any of the other cases: Klopstock and Kowarsky²¹ assert that the urine should be examined in as fresh a condition as possible, as diacetic acid rapidly decomposes into $C.O_2$ and acetone. As the specimens of urine for examination were usually obtained from the total twenty four hours collection in the foregoing cases, it is probable that diacetic acid might have been found more frequently, and also in the urine of other cases, if the specimens could always have been obtained in as fresh a condition as possible.

Though acetone was present in the urine of all the fourteen post anaesthetic cases examined, it was not tested for in Case I. till the third day, when in addition diacetic acid was present; acetonuria persisted till the eighth day in this Case.

If the days of the puerperium on which acetone was present be analysed, it will be seen that:-

On the first day, eight of the thirteen cases examined shewed acetone.

On the second, twelve of the thirteen cases examined shewed acetone, and

On the third, nine of the fourteen cases examined shewed acetone.

Acetone was demonstrable on the first day only in Case XI.

Acetone was demonstrable on the second day only, in Case X.

In four cases (IV. V. VIII. and XIV.) it was present on the first three days inclusive.

In brief, acetone was present as a rule on two of the first three days of the puerperium, in twelve of the fourteen cases examined, if we include Case I: or in 85.6 per cent.

Couvelaire and Scholten²², state that acetone is demonstrable in the urine of all parturient women for the first two days of the puerperium; they state further that the amount is increased, in 94 per cent of the cases examined: the acetonuria disappears by the third day, and they assign its presence as being due to the increased muscular activity incident to parturition.

Boston²³, states that under normal conditions acetone may appear as a faint trace in the urine, the amount varying in the daily output, from .08 to .25 mg.

Klopstock and Kowarsky²¹ point out that the ordinary Sodium Nitroprussiate Test (Legal's) cannot detect amounts of acetone under .8 millegramme; they consider that the Iodine Potassium Iodide reaction of

Lieben is much more delicate, but that neither of these can detect the faint trace of acetone present in normal urine, to demonstrate which the still more delicate quantitative test is necessary, as recommended by Hoppe-Seyler²⁴.

Beesly⁸ proved acetone to be present in the urine of 200 post anaesthetic cases, by means of the colour tests in addition to the quantitative estimation.

In every case when acetone was present by Legal's Sodium Nitroprussiate Test, the colour evidenced was at first purple, (in Case I. being a deep blackberry colour): which usually faded, if acetone was present on more than one day, to a fainter purple or even to a horse chesnut hue, in Cases VIII. and XIV. on the last day.

It has been shewn by control quantitative tests, that when the foam in the Test tube is of a horse chesnut colour, acetone is present although in slight degree.

The reaction of the urine in the nineteen cases, was markedly acid on the first day, this gradually became less acid.

The acidity was rapidly influenced by the administration of Sodium Bicarbonate, except in Case I.

when the drug was initiated on the third day and the urine did not become alkaline till the eighth day.

Sodium Bicarbonate was only used in the puerperia of the first five Cases and in Case IX.

The deposit. Blood corpuscles were always present, and altered fragments of fibrin.

The crystals like Tyrosin occurring on the third day in Case I. have already been noted; it should be mentioned that Leucin was not found present.

Uric Acid was present in ten of the fourteen cases, or in 71.4 per cent; when it was in the form of sheaves, spears, and needles, as a rule.

In two of the five cases without chloroform, uric acid crystals were also present.

Amorphous urates were present in every case examined, in varying degree; the deposit being most marked on the first and second days.

Triple phosphates were observed in Cases IV. and XI. Oxalates were never found present.

THE TESTS EMPLOYED.

The tests for acetone employed were:-

LEGAL'S TEST:- To a specimen of the urine add from three to five drops of a freshly prepared solution of Sodium Nitroprussiate, render alkaline with a few drops of Caustic Soda, when a ruby red colouration will appear: if this red solution is supersaturated with acetic acid the red colour passes into crimson, or

even to a dark blackberry purple: if this be shaken the foam will shew the same colour, the depth of colour depending on the amount of acetone present.

LIEBEN'S TEST:- To a specimen of urine add five to ten drops of Lugol's Iodine potassium iodide solution and sodium hydrate. In the presence of acetone, iodoform is produced which may be easily recognised by its odour and its crystalline forms.

The urine to be examined was first treated with Legal's test; if this was negative, or doubtful, then Lieben's was used.

GERHARDT'S TEST FOR DIACETIC ACID. A solution of Ferric Chloride is added to a specimen of urine. If diacetic acid is present a red colouration is produced.

TEST FOR LACTOSE. A specimen of urine is boiled for three or four minutes with a large quantity of lead acetate: in the presence of lactose the solution becomes brownish yellow; then ammonia is added to the hot solution. An intense brick red colour is first produced which throws down a copper red precipitate and then becomes colourless.

The quantitative estimation of urea was performed by the ureometer of Doremus.

The quantitative estimation of acetone was not carried out owing to the time required to perform the method described by Hoppe-Seyler, which takes on an average four hours to complete.

DISCUSSION OF THE CLINICAL SIGNS OBSERVED

IN THE FOURTEEN POST ANAESTHETIC CASES.

In four of these Cases (I. II. III. IV.) there were certain definite toxic phenomena noted, which

were present within the first few days of the puerperium.

There was no apparent disease present of the heart, lungs, liver, or kidneys, previous to the labour.

Case V. gave evidence of a long standing affection of the hepatic system and accordingly is not grouped with the above cases; although headache, nausea, and jaundice were apparent on the first day of the puerperium.

All the fourteen cases shewed acetonuria to be present during the puerperium: and as noted above, the first four cases had in addition, toxic phenomena of varying degrees of intensity.

Accordingly 100 per cent evidenced acetonuria and 28.5 per cent had super-added toxic symptoms.

The signs and symptoms observed in, and complained of by the cases, were:-

(1) Headache, (2) Nausea, (3) Vomiting, (4) The smell of acetone in the breath, and its sweet taste in the mouth, (5) Pupil changes, (6) Jaundice of varying degree and (7) Drowsiness, restlessness, and delirium.

HEADACHE. This was frontal in all the cases; it was most intense in Case I., not so severe in Case II., and of lessening degrees of intensity in Cases III. and IV. In the first case, the headache was not complained of until the ^{end of the first} ~~second~~ day: in the other cases it began about twelve hours after the cessation of labour.

NAUSEA; was apparently concomittant with the headache; the degree of nausea was very similar to the so-called "sick headache" variety.

VOMITING. Was noted in the first two cases only: it was never haemorrhagic.

In Case I., it did not begin until eleven p.m. on the third day; when it persisted throughout the night with short intermissions, and continued well into the fourth day.

The second case vomited twice between four and six a.m. on the second day, more than twenty four hours after the cessation of labour.

THE SMELL OF ACETONE IN THE BREATH, was only evidenced in the first case; it was first appreciable on the third day; coincident with this, the patient complained of a sweet taste in her mouth like apples. When the poisoning symptoms were at their height on the fourth day, the acetone odour was apparent some little distance away from her mouth.

The taste of chloroform occasionally complained of by one or two of the cases, is not to be confused with the true acetone taste.

CHANGES IN THE PUPILS, were noted in Case I. only, when they were widely dilated on the fourth day: the dilatation persisted till the "storm" of clinical phenomena had subsided.

JAUNDICE. This was noted in varying degrees of intensity in the first four cases: it was also apparent in Case V.

In Case I., the jaundice was first apparent on the second day, being most marked on the cheeks, the sides of the nose, and in the conjunctivæ: as the degree of toxæmia increased in severity, the colour was most pronounced on the fourth day, when the skin was dry and intensely yellow all over the body, but was most marked on the face: the general jaundice faded rapidly after the symptoms subsided, but the conjunctivæ were not absolutely clear until fifteen days later.

In Case II., the degree of jaundice was slight in comparison, appearing on the second day and being most marked in the conjunctivæ: the skin over the neck and chest shewed slight evidence of a yellow stain, after careful examination in a good light.

In Cases III. and IV. the jaundice was very slight; in the former being apparent round the eyes and in the conjunctivæ, in the latter only being definite in the conjunctivæ.

It should be here noted that all these cases were dark haired women, with the sallow white skin peculiar to their type: hence, it was no easy matter to determine whether jaundice was present on the body or not, except in Case I.: and also in Case V., in which

instance the degree of yellow was easily recognised, by reason of its having been noted before in the past.

DROWSINESS, RESTLESSNESS, AND DELIRIUM. These conditions were only observed in Case I.

On the second day the patient was restless and drowsy in the evening: during the subsequent night the restlessness was so marked that the nurse tied the thighs together to protect the perineal stitches.

The drowsy condition was not the ordinary drowsiness associated with fatigue; she complained of wanting to sleep, and would lie quiescent with closed eyes, and perhaps dosed for a short time: then she would suddenly toss her arms about, turn in bed, and try lying in another position: as she became worse the restless state increased, until she was markedly delirious on the third night, crying out and talking incoherently: when she was seen on the fourth day she responded when spoken to, but could not talk rationally.

Whenever the vomiting became less persistent the mental condition rapidly improved, and by night time of the fourth day she was quite clear mentally, but could only recall the events of the past few days as a bad dream.

If the toxic symptoms observed in the first four cases be compared, it is of course evident that the

degree of intensity is most marked in Case I., much less evident in Case II., and again even less in Cases III. and IV. In all four, headache, nausea and jaundice were apparent with marked degrees of difference, the last three cases being cases in "miniature" as it were.

It would appear as if the signs of poisoning in Case I. had been held back in some unexplained way, until the second day, when they rapidly progressed to their height on the fourth; the first day of this case being "the calm before the storm".

Headache, Nausea, and Jaundice in Case I. were supplemented by drowsiness, restlessness, persistent vomiting, the smell of acetone in the breath, and eventually delirium with widely dilated pupils - all differences, admittedly: but differences in degree.

Hence it is reasonable to infer that the varying signs in each case were toxic manifestations of the chloroform, in different degrees.

Now the first case affords an example of a high degree of poisoning which may be likened to the most intense variety of delayed chloroform poisoning which frequently ends in death: Cases II. III. and IV. apparently shew lessening degrees of intensity.

Hence it is probable that - unless the last three cases had been approached with the view of noting any clinical phenomena which might be attributed to the chloroform, such as headache, nausea, or jaundice, - these symptoms observed might have been assigned to some other cause, if they had been noted at all.

This point should be emphasised, as it has been already by Stiles and MacDonald, namely:- that all degrees of intoxication may be present after chloroform anaesthesia, and accordingly the condition must not be mistaken for delayed shock or sepsis, unless there is some definite evidence of their presence.

THE DIAGNOSIS.

The possible true diagnosis of Case I. did not suggest itself until the third day of the puerperium; and unless the smell of acetone had been noted in the breath, and the writer had previously seen cases of post anaesthetic acetonuria of toxic degree, it is probable that the condition might have been missed, or might have been attributed to possible acute yellow atrophy of the liver, or to septicaemia.

Because parturient women tolerate chloroform safely is no reason why symptoms of poisoning may not exist which may be directly attributable to the anaesthetic -

although such cases as Case I. are admittedly rare.

It is probable that in all cases of labour after Chloroform, there are some after effects which are not usually recognised, e.g. acetonuria; and further, when headache or nausea occurs early during the puerperium, these symptoms may be due to the "delayed action" of the chloroform, unless there is some definite reason present which could prove the contrary.

The writer observed two cases in a Maternity Hospital some years ago, which had both been safely delivered under chloroform anaesthesia: in both, there was marked headache, nausea, and vomiting of slight degree on the second day; they both recovered uneventfully. When the House Physician was asked to give some reason why these cases should react so differently to the usual run of labours, the answer given was, "that the signs observed might be due to the shock of the labour, or were possibly due to some slight septic change". If the golden rule of aiming at a true and comprehensive diagnosis be followed, the above answer is not satisfactory; for though the "shock of labour", or "slight septic changes", may be occasionally met with in the ordinary examples of parturition with chloroform, the fact remains that the anaesthetic, which also might have been responsible,

was not mentioned.

These remarks are not made with the view of adversely criticising the administration of chloroform in labour; far from it, the writer always employs reasonable chloroform anaesthesia in all cases when possible. But it should be pointed out that possibly the attitude of the Medical Profession towards Chloroform in parturition has become somewhat overconfident: this is no doubt because of the well known immunity of the parturient woman. As a case in point, the probability of the chloroform being the cause of the dangerous phenomena in Case I., never suggested itself to the writer until acetone was apparent in the breath.

As has been emphasised before, the symptoms of acute yellow atrophy of the liver are very similar to those occurring in severe cases of delayed chloroform poisoning, though the reason of this is not yet explained.

In Anning's case (see page 21.) no explanation is made to account for the signs observed, except by the conception of acute yellow atrophy of the liver; in fact he concludes with the remark, that "Rolleston states that chloroform narcosis is on rare occasions followed by acute yellow atrophy".

This may be so, but the writer ventures to suggest that delayed chloroform poisoning ought to be brought forward as the possible cause in all such cases as Anning's, unless definitely proved to the contrary.

Further, the "acute yellow atrophy element" in such cases may be merely a manifestation of the intensity of the delayed chloroform poisoning; and in the present state of our knowledge, one cannot exactly determine either condition from the diagnosis, until the evidence of a post mortem examination is obtained.

Accordingly, although a correct diagnosis is at present impossible, signs and symptoms of poisoning whenever apparent should at once be recognised, and treated on ant-acid lines; this may be of vital importance, for while the diagnosis is being attempted, the degree of acid intoxication may be advancing rapidly, and the patient's chance of recovery lessened by the delay.

Case I. has impressed the following fact on the mind of the writer, namely that all degrees of poisoning after chloroform may be observed in cases of parturition, just as in post operative cases.

This might influence the diagnosis when any question of sepsis should arise.

The ratio of the pulse to temperature in cases of

sepsis, differs from the condition in Delayed Chloroform poisoning, when the pulse may be markedly accelerated, while the temperature is only slightly raised if at all.

For example, if on the ^{first or} second day of the puerperium following a labour "with chloroform", the pulse rate should be accelerated, with accompanying headache, and nausea, the question of the early onset of sepsis would be apparent: this probability would be further strengthened if there had been a lacerated perinaeum, or some instrumental interference during the labour. But if there had been no rigors, and the lochial discharge shewed no quantitative nor qualitative changes, and no tenderness was present over the lower abdomen, in addition to a temperature only slightly raised if at all, the writer ventures to suggest that some other cause than sepsis would have to be looked for, and that as ^a temporary measure ant-acid treatment might be adopted.

In other words such phenomena as the foregoing (or such as occurred in Cases II. III. and IV.) might be rightly assigned to the delayed action of the chloroform.

It is admitted that a septic condition would not be negatived by the above, the further progress would of course have to be carefully watched, but is is very

important that the delayed action of chloroform should be borne in mind.

PROGNOSIS.

The amount of Chloroform administered and the duration of the anaesthesia would not appear to have any effect on the prognosis of a case; provided that the anaesthesia was "obstetrical", i.e. intermittent and of light intensity between the pains.

If acetonuria should be present during pregnancy, and at parturition chloroform be administered, it is probable that the prognosis would not necessarily be affected; so long as digestive disturbances had not been present antecedent to the labour.

Beesly⁸ has proved, that the usual risks of anaesthesia were not influenced by pre-existing chronic acetonuria.

The prognosis of a case which shewed violent symptoms of delayed chloroform poisoning would depend on various factors:- the existence of previous disease, and in addition as to whether the state was one of acute yellow atrophy of the liver.

It follows that if acid intoxication should arise during the puerperium of a woman who is already in a state of auto intoxication, the outlook would be more serious.

Further, if renal insufficiency should be present before the onset of delayed chloroform poisoning, the extra excretion of the products of the acid intoxication would tell heavily on the already embarrassed renal system.

But an extenuating circumstance should be added to the prognosis of all cases of doubtful acid intoxication or acute yellow atrophy of the liver, namely, that in such a case until ant-acid treatment has had a fair trial, all has not been done that is possible for the patient; and in addition some responsible lay member of the household should be told of the possibility of the poisoning yielding to ant-acid treatment, when the grave warning is given as regards such cases.

TREATMENT.

It is essential that the parturient woman should be in as satisfactory a state of health as possible before she undergoes her labour; and as chloroform is used in practically all such cases nowadays, it is very necessary that her "state of resistance" should be highly efficient, so as to more ably neutralise the acid-forming elements caused by chloroform.

The rare cases of Post Anaesthetic Acid Intoxication which are very similar to Acute Yellow Atrophy of the

Liver, give very little warning of their onset: in fact if the theory of Idiosyncrasy is granted, no one can guarantee that a case will not shew subsequent signs of Delayed Chloroform Poisoning, which may be so grave as to jeopardise life itself.

Hence it is very important that the rare possibility of all degrees of Delayed Chloroform Poisoning occurring after Labour, should be recognised and treated.

As has been already stated on page 23 the writer determined to adopt a routine line of prophylactic treatment in cases of pregnancy which shewed undue digestive disturbance with obstinate constipation, or acetonuria, during the later months: and further, it was resolved to modify the diet of pregnant women in general during the last month whenever possible, with a view to their subsequent reaction to chloroform.

The above routine procedure was carried out in the last nine cases (VI. - XIV); when they all shewed acetonuria, but with no accompanying toxic symptoms during the puerperium of any case.

Hence the treatment of parturient women should be Prophylactic with a view to subsequent chloroform anaesthesia; as well as "active" during the puerperium

if occasion should arise.

PROPHYLACTIC TREATMENT. The Prophylaxis should consist of the ordinary routine regulation of the bowels, more especially during the latter months of pregnancy, by the use of suitable aperients, so as to ensure of a daily evacuation.

The diet should be modified in all cases, especially towards the last month of pregnancy, when less fat should be ingested, and also less proteid in the form of meat: on the other hand there should be an increase in the intake of carbohydrate.

The above procedure as to the diet, was suggested by the treatment of acetonuria in general, as recommended by Langdon Brown⁴ and others; the rationale of which is to diminish the amount of fat in the food, on the grounds that the acetone bodies are chiefly derived from fat; and further, that a deficiency of carbohydrate causes defective oxidation in the tissues (Liver and Muscles), and hence, there is defective metabolism of fat with the formation of the acetone bodies.

If digestive disturbances should exist apart from constipation, or if acetonuria be present, a course of sodium bicarbonate should be exhibited, as recommended by Beesly⁸ in the pre-anaesthetic treatment of children.

ACTIVE TREATMENT DURING THE PUERPERIUM. If

Acetonuria be present with no Toxic symptoms, no Ant-acid treatment is necessary.

If a slight degree of poisoning should be present, which may be attributed to the chloroform, such as:- Headache and Nausea with Acetonuria; Sodium Bicarbonate (40-60 grains) should be administered every two or three hours, in conjunction with a free fluid diet of milk, milk and soda, Bengers Food etc.

If the symptoms subside the alkali may be withheld on the second day, when the urine should be neutral or alkaline, or only slightly acid.

But if there should be a severe degree of Acid Intoxication, every means in our power must be taken to counteract this, by the administration of alkalies and by free elimination:- For example, the skin should be made to act freely by means of blankets and hot bottles, or if necessary by vapour^{or hot water} baths: a smart purge should be given in addition: and sodium bicarbonate administered in drachm doses frequently.

If the vomiting is persistent, lavage may be necessary; and the procedure as recommended by Spriggs²⁵ should be followed:- After the lavage, a solution of sugar being introduced into the stomach: or the method recommended by Wallace and Gillespie²⁶

may be adopted, which consists of the exhibition of Glucose in half ounce doses by the mouth (whenever possible.)

The bowels should be encouraged to act freely by copious enemata, and this may be followed by rectal injections of sodium bicarbonate solution, consisting of one ounce to the pint of hot water.

If the acid intoxication should persist, the subcutaneous infusion of sodium bicarbonate in two per cent solution made up in normal saline should be carried out; and if no improvement follows it may be expedient to administer the saline intravenously.

Coincident with the Ant-acid treatment, the general condition of the patient should be carefully noted especially as regards the pulse and temperature: and the further possibility of a superadded septic condition watched for.

SUMMARY.

The questions formulated on page I. may now be answered on the admittedly small basis of the results obtained in fourteen cases of parturition with chloroform:-

(1) Whether Acetonuria was present before and after the labour.

Acetonuria was present before the labour in one

case only (Case V.): it was present after the labour in all the cases.

(2) Whether any pre-existing condition of the Digestive, Circulatory, Respiratory, or Renal Systems was present, which might predispose to chloroform poisoning.

There was no apparent pre-existing condition of any of these systems, which might have predisposed to Delayed Chloroform Poisoning except the presence of constipation in Cases I. II. III. and IV.

Case V. gave a previous history of gall stones which might have been a predisposing factor; but the subsequent post anaesthetic signs and symptoms were of a very slight degree: it should be noted that in Case V. prophylactic measures were stringently enforced.

(3) Whether any signs of poisoning supervened which might be directly attributable to the chloroform anaesthesia.

There were definite signs of poisoning of varying degree in Cases I. II. III. and IV. which were apparently attributable to the action of the chloroform.

Case V. is not included in these Cases.

(4) Whether any train of symptoms existed which might help to make the diagnosis of chloroform poisoning distinct from sepsis.

The only signs which apparently helped the differential diagnosis were:- the early onset of the toxic phenomena, and the ratio of the pulse to the temperature (when the pulse was most perceptibly

increased, the temperature being only slightly raised.)

The diagnosis chiefly depended on the absence of the following signs of sepsis, namely:- Rigors, alterations in the lochial flow, tenderness over the lower abdomen, and a swinging temperature.

(5) Whether any early train of symptoms existed which might indicate ant-acid treatment.

Headache and Nausea, and Jaundice of varying degree occurred in Cases I. II. III. IV. and V. and in consequence ant acid treatment was at once adopted within twenty four hours of the cessation of labour.

The diagnosis of acid intoxication was not made in Case I. until the third day, and in consequence treatment on ant acid lines was not adopted till then.

THE PUERPERAL URINE.

As regards the urine of the puerperium, following cases of parturition with chloroform the conclusions are:-

- (1) That acetonuria may be demonstrable by the ordinary qualitative tests, during the first few days.
- (2) That the daily amount of urine excreted just after labour is less than the normal as a rule.
- (3) That the excretion of uræa is at first increased.
- (4) That it is probable that chloroform may tend to increase the excretion of urea.

(5) That lactosuria may be present coincident with the secretion of milk.

(6) That occasionally uric acid crystals, and practically always amorphous urates are present in the urine of the first two days of the puerperium.

Finally, the following general conclusions may be drawn with reference to parturition with chloroform:-

(1) That obstetrical anaesthesia is indicated in all cases of parturition occurring in healthy women: but that the possibility of various degrees of Delayed Chloroform Poisoning should be recognised.

(2) That Post-anaesthetic Acetonuria may be present during the puerperium without toxic symptoms.

(3) That on rare occasions, a grave condition of acid intoxication may result, which might cause death: and further, that at present the signs of intoxication are not clinically diagnostic from those of Acute Yellow Atrophy of the Liver.

(4) That ^{Severe} acid intoxication occurring in previously healthy women may be explained by the assumption of idiosyncrasy.

(5) That occasionally a mild degree of intoxication may occur, which might be erroneously diagnosed as being due to delayed shock or sepsis.

(6) That Jaundice may be present, with no apparent

change of the liver dulness, and associated with a varying degree of "malaise".

(7) That early and immediate "Ant-acid" treatment is indicated in all cases of acid intoxication, apart from acetonuria alone.

The object of this paper is to point out that Post Anaesthetic Acetonuria may be present after a labour in which Chloroform has been used: and further, to emphasise the fact that varying degrees of acid intoxication may occur - from the grave condition which closely resembles acute yellow atrophy of the liver, to the mild condition of Acetonuria with only slight headache and nausea.